

that emaciation is not pronounced, that fever is lacking, the pulse-rate is normal, and that there is obtained clear resonance above the clavicle, etc., should be counted against tuberculosis. The more severe lesions in which pulmonary abscess remains after influenza the diagnosis may be made along the same lines as in bronchiectasis, and in addition note should be made of the fact that the sputum is abundant, at times fetid, the fever continuous or undulating, the pain in the chest and the physical signs, all of which combine to clear up the diagnosis. If several examinations of the sputum fail to disclose tubercle bacilli the diagnosis may be safely made. Leukocytosis, with an increased proportion of polymuclear cells have the same significance.

STUDY OF A CASE OF UNEXPLAINED LOW CARBON DIOXIDE COMBINING POWER OF THE BLOOD.

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THE following case is reported because of the persistent occurrence of a low carbon dioxide combining power of the blood and an increase in the rate and depth of respiration in an individual in whom no adequate explanation for these phenomena could be discovered. The patient's chief complaint was shortness of breath and a feeling that his heart beat fast; the abnormal respiration was sufficiently marked to attract immediate attention when one approached the patient. It was this observation which led to the further special study of the case as well as the fact that the physical examination revealed no cause for the obvious increase in respiratory frequency; nor did the routine laboratory studies reveal any of the disease conditions which we are accustomed to expect as leading to dyspnea. No justification for a diagnosis of cardiac, pulmonary or renal disease was found, nor was there any evidence of diabetic acidosis; in fact, no evidence of disturbed acid-base equilibrium other than the constantly low plasma carbon dioxide combining power. There was no fever.

In brief, the patient presented an unusual picture which we are unable to explain, and we are reporting the facts without attempting

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to draw any conclusions. Undoubtedly a reasonable explanation exists, to become apparent only after further facts are known.

CASE REPORT.—G. H., male, aged sixteen years, entered the University Hospital on February 9, 1920, complaining of shortness of breath and a feeling that his heart beat fast. The history in brief was as follows: He was in perfect health until about five weeks before admission, when he had run for over an hour without any previous training. He was accompanying a boxer who was training, and they ran "at least five miles." There was no special discomfort at the time, but three days later he became conscious of the beating of his heart. A week later he had an acute attack of dizziness and vomiting, which continued for two days. He stated that he had not been well since the overexertion in running, and he claimed to have lost ten pounds during the two weeks prior to admission. Previous medical history was negative and there was nothing of importance in the social or family history.

The patient was a healthy looking individual, obviously a mouth-breather. It was noted at once that the breathing was more rapid and deeper than normal; the rate varied between 24 and 32 to the minute. The hands and fingers were somewhat cyanosed, but not markedly so; the blood-pressure was low: systolic 96, diastolic 68. Definite tonsillar enlargement was present and also a slight general adenopathy. In all other respects the physical examination was negative. Repeated examinations of the heart by several examiners and by fluoroscopy and electrocardiography revealed no alteration from the normal. The lungs were normal by physical examination; roentgenograms revealed no pulmonary pathology nor any mediastinal abnormality. Neurological examination was negative.

The routine laboratory examinations were as follows: Blood count: Erythrocytes, 4,410,000; leukocytes, 7000; hemoglobin, 71 per cent. Differential count: Polymorphonuclear neutrophils, 73 per cent.; lymphocytes, 22 per cent.; large mononuclears, 4 per cent.; transitionals, 1 per cent. The serologic test for syphilis was negative. As is our custom, specimens of urine were sent to the laboratory on the first, third and fifth mornings after admission. These showed striking variations in the concentration, albumin content and microscopic findings. Such changes are familiar to all hospital physicians and result at least in part from the increase in fluid intake incident to hospital care.

Color.	Sp. gr.	Albumin.	Microscopic.
1. Dark amber	1029	Heavy cloud	Light and dark granular casts.
2. Amber	1015	Very faint trace	0
3. Straw	1007	0	0

In all other respects the examinations were negative. On the day of the third specimen the phthalein elimination was 60 per cent.

in two hours and the blood-urea nitrogen was 15 mg. per 100 c.c. of blood.

Other special examinations which should be mentioned included a negative examination of the eye-grounds and negative roentgenograms of the pituitary region. Roentgenograms of the teeth revealed an abscess at the root of a molar tooth.

The following table will detail the further study of the case:

1920	Sod. bi-carbon., gm.	Blood plasma, CO ₂ vol., per cent.	Urea N., mg per 100 c.c.	Urine.				
				Amount, in c.c.	Specific gravity.	Reaction.	NH ₄ N.	Titratable acidity, N/10 NaOH
Feb. 14	5	29.0	15					
15	20							
16	5	42.5						
17	2840	..	Acid	0.30	249.2
18	2540	..	Acid	0.24	223.1
19	..	48.2	..	3000	..	Acid	0.44	368.0
20	..	53.0	15	2800	..	Acid	0.59	396.0
21	..	52.0	..	4000	..	Acid	0.59	747.0
22	5000	1.004	Arid	0.26	571.0
23	..	47.0	..	4760	1.005	Acid	0.36	786.0
24	..	42.0	..	3850	1.008	Acid	0.36	857.0
25	..	47.0	..	3620	1.008	Acid	0.36	
26	..	46.0	..	3400	1.004	Acid	0.31	439.0
27	20	46.0	Spec. lost	
28	20	48.0	..	3870	1.007	Arid	0.22	104.5
29	20	3160	1.008	Acid	0.20	104.5
Mar. 1	20	50.0	..	2310	0.008	Acid	0.25	
2	25	54.0	..	3920	1.008	Acid	0.18	170.5
3	20	59.0	12	2660	1.010	Sl. alk.	0.12	Neutral
4	25	55.0	..	2230	1.011	Sl. alk.	0.18	120.0
5	1910	1.008	Acid	0.27	176.0
6	..	49.0	Spec. lost	
7	2320	1.009	Acid	0.42	288.0
8	2680	1.010	Acid	0.46	171.0
9	..	54.0	..	1780	1.010	Acid	0.21	363.0
10	940	1.016	Acid	0.31	284.0
11	..	55.0	..					
12	..	55.0	12					
15	..	52.0	..					
17	..	43.0	..					
18	..	48.2	12					
22	..	47.9	..					
23	..	47.0	..					
27	..	49.0	..					
30	12					
April 1	..	53.0	..					
6	..	52.0	..					
14	..	53.0	12					
June 1	..	55.0	17					

Interest was aroused by the report that the plasma carbon dioxide combining power was reduced to 20 volumes per cent. and a corresponding low alveolar carbon dioxide tension. No observations

were made to determine the influence of the low CO_2 combining power of the plasma on the power of the hemoglobin to combine with oxygen.

The examination of the data contained in the protocol together with an absence of both a ketonuria and a glycosuria and the presence of a normal blood sugar immediately excluded diabetes as the cause of the low CO_2 combining power of the plasma.

The examination of the urine for lactic acid was negative for any abnormal production of this acid.

We feel that nephritis, as has been previously stated, can be eliminated by the constant absence of urea retention and the normal phenolsulphonephthalein elimination. Albumin and casts were found in the first examination of the urine, but all subsequent examinations were negative. Although a marked polyuria and a corresponding low specific gravity were present for a number of days the kidneys were able to concentrate, as was shown by the first urinary examination when the specific gravity was 1029, and on April 27, 1920, when the intake of fluids was greatly restricted, with a result that the output was reduced to less than 500 c.c. per twenty-four hours and the specific gravity raised to 1029. We do not feel that the polyuria is especially peculiar to the condition because of the great ingestion of fluids, particularly water and milk. The striking feature of the case is the low CO_2 combining power of the plasma with the constant normal output of ammonia nitrogen and a normal titratable acidity, except from February 20 to February 24, when the titratable acidity was above normal. The cause of the increased titratable acidity at that time is unexplained. Although a total of 30 gm. of bicarbonate of soda was given between February 14 and the morning of February 16, we feel that it did not influence the ammonia nitrogen and titratable acidity on the subsequent days, particularly in view of the continued low CO_2 combining power of the plasma.

Henderson¹ has shown that hyperpnea may cause a marked lowering of the carbon dioxide combining power of the plasma with a normal urinary ammonia and titratable acidity. We investigated the possible application of this idea to our patient, assuming that the rapid, deep breathing was due either to an oversensitive respiratory center or stimulation of the center by a neoplasm, perhaps of the pituitary, in view of the polyuria. Negative roentgenographic findings excluded a pituitary tumor as the cause. If due to over-ventilation then the depression of the respiratory rate should raise the blood CO_2 combining power with no ill-effects, such as would result if due to the usual clinical causes of acidosis. To test this possibility, morphin sulphate was given hypodermically from

¹ Jour. Biol. Chem., 1918, xxxiii, 345, 355, 365.

February 18 to February 21. By February 21 the respiratory rate was reduced to 16 per minute, and during this period the blood CO_2 reached its highest figure, except early in March, when the high figure was due to the administration of sodium bicarbonate. Unfortunately the specimen of blood which was taken just before the beginning of the administration of morphin sulphate was lost, due to an accident. After the effects of the morphin sulphate had ceased the blood CO_2 again fell. No ill-effects on the patient were observed during this experiment.

Although these experiments seem to admit the possibility of overventilation as the cause of this unusual phenomenon in the patient, we do not feel that the data at hand is sufficiently conclusive to permit this or any positive diagnosis. The sensitiveness of the respiratory center was tested on March 3, after the blood CO_2 had been raised to normal, by the administration of sodium bicarbonate from February 27 to the date of the experiment. The method employed was to have the patient breathe normal air and then air containing 5.2 per cent. of CO_2 , recording the rate and depth of the respirations on a kymograph. It was found that the breathing of air containing 5.2 per cent. of CO_2 caused a turn-over of air twice that observed before the CO_2 was increased. This is considered a normal reaction for that concentration of CO_2 .

Other possible explanations have to be considered, but none appear satisfactory. Hysterie tachypnea, such as was described by S. Weir Mitchell, and such as is not infrequently observed clinically, is apparently ruled out by the persistence in this case of the respiratory rapidity during sleep. The acute attack of indigestion with vomiting can scarcely be considered prolonged enough to have disturbed the patient's acid-base equilibrium for so long a period, nor were the alterations in this case those seen after vomiting, starvation or in "cyclic vomiting," such as ketonuria, etc.

EMPYEMA THORACIS: AN ANALYSIS OF FIFTY-SIX CASES IN THE INDIANA UNIVERSITY HOSPITAL.*

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EMPYEMA literature of the last two years is voluminous enough to give the average reader mental anorexia. The subject has necessarily been clouded with uncertainty, so rapid have been the changes in conception of both pathology and treatment. The aim of this

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